OX40L/OX40 axis impairs follicular and natural Treg function in human SLE

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Résumé en anglais	Tregs are impaired in human systemic lupus erythematosus (SLE) and contribute to effector T cell activation. However, the mechanisms responsible for the Treg deficiency in SLE remain unclear. We hypothesized that the OX40L/OX40 axis is implicated in Treg and regulatory follicular helper T (Tfr) cell dysfunction in human SLE. OX40L/OX40 axis engagement on Tregs and Tfr cells not only specifically impaired their ability to regulate effector T cell proliferation, but also their ability to suppress T follicular helper (Tfh) cell-dependent B cell activation and immunoglobulin secretion. Antigen-presenting cells from patients with active SLE mediated Treg dysfunction in an OX40L-dependent manner, and OX40L-expressing cells colocalized with Foxp3+ cells in active SLE skin lesions. Engagement of the OX40L/OX40 axis resulted in Foxp3 downregulation in Tregs, and expression in SLE Tregs correlated with the proportion of circulating OX40L-expressing myeloid DCs. These data support that OX40L/OX40 signals are implicated in Treg dysfunction in human SLE. Thus, blocking the OX40L/OX40 axis appears to be a promising therapeutic strategy.
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